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16. ABSTRACT

Selected technical literature on biological and ecological effects of mercury compounds on marine and estuarine biota is reviewed. Potential and actual hazards to public health through marine vectors are considered. Within this framework, approaches for establishing mercury contamination standards in saline environments are presented.

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ENERGY AND ENVIRONMENTAL STRESS IN AQUATIC SYSTEMS

Selected papers from a symposium held at Augusta, Georgia
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PREFACE

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MERCURY CONTAMINATION STANDARDS FOR MARINE ENVIRONMENTS

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Environmental Protection Agency, Environmental Research Laboratory, Narragansett, Rhode Island

ABSTRACT

Selected technical literature on biological and ecological effects of mercury compounds on marine and estuarine biota is reviewed. Potential and actual hazards to public health through marine vectors are considered. Within this framework, approaches for establishing mercury contamination standards in saline environments are presented.

Toxicological aspects of mercury and mercury compounds in coastal and offshore environments as a result of anthropogenic or natural processes have been extensively reviewed elsewhere (D'Itri, 1972; Friberg and Vostal, 1972; Gavis and Ferguson, 1972; Harriss, 1971; Holden, 1973; Jernelöv, Landner, and Larsson, 1975; Keckes and Miettinen, 1972; Newberne, 1974). Most of these authorities agree on five points. First, forms of mercury with relatively low toxicity can be transformed into forms with very high toxicity through biological and other processes. Second, uptake of mercury directly from seawater or through biomagnification in marine food chains returns mercury to man in concentrated form. Third, mercury uptake may result in genetic changes. Fourth, elevated levels of mercury in some marine fishes, such as tuna or swordfish, emphasize the complexity of both natural mercury cycles and man's impact on those cycles. Finally, man's use of mercury should be curtailed because, in contrast to some other pollutants, the difference between tolerable natural background levels of mercury and harmful levels in the environment is exceptionally small.

This paper considers recent material on mercury effects in saline waters and recommends useful criteria and promising research approaches for incorporating mercury contamination standards that will protect marine products of commerce, their food organisms, and their predators, including man.

HISTORICAL REVIEW

Laboratory Studies

Comparative Toxicity, Survival, and Biotic and Abiotic Modifiers

In general, salts of mercury and its organic compounds have been shown in short-term bioassays to be more toxic to marine organisms than are salts of other heavy metals. To oyster embryos, for example, mercury salts were more toxic than were Ag, Cu, Zn, Ni, Pb, Cd, As, Cr^{+6}, Mn, or Al (Calabrese et al., 1973); to clam embryos, mercury was the most toxic metal tested, followed by Ag, Zn, Ni, and Pb, in that order (Calabrese and Nelson, 1974). Toxicity bioassays of 168 hr duration with salts of Hg, Cd, Cr^{+6}, Ni, and Zn on adults of representative marine fishes, crustaceans, bivalve and gastropod molluscs, annelids, and echinoderms confirmed that mercury was consistently the most toxic metal in this series (Eisler and Hennekey, 1977). Similar results are reported for marine algae (Berland et al., 1976); marine fungi (Schneider, 1972); sea urchin eggs (Kobayashi, 1971); larvae of marine molluscs and crustaceans (Connor, 1972); some species of marine polychaete worms (Reish et al., 1976); freshwater annelids, insects, and gastropods (Rehwoldt et al., 1972); crustaceans (Cabejszek and Stasiak, 1960); and fish (Weir and Hine, 1970).

Acute toxicity values of mercury compounds for various marine species are summarized in Table 1. These values are similar to those reported for freshwater groups (McKim, 1977). Differences in species resistance to mercury compounds (Hendrick and Everett, 1965; Schweiger, 1957; Wisely and Blick, 1967) may account for some of the variability in test results shown in Table 1, but many additional factors are known to affect or modify the survival time of mercury-stressed marine biota, sometimes by one order of magnitude or more. Abiotic modifiers include the chemical form of mercury administered (Boetius, 1960; Boney, 1971; Boney and Corner, 1959; Boney, Corner, and Sparrow, 1959; Corner and Sparrow, 1957; Ellis, 1947; Fang, 1973; MacLeod and Pessah, 1973; Middaugh and Rose, 1974); cations other than mercury present in the medium (Barnes

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TABLE 1

ACUTE TOXICITY OF MERCURY COMPOUNDS TO VARIOUS SPECIES OF MARINE BIOTA*

Species	Concentration	Effect	Reference
Algae			
18 spp.	10—25	LC_{100} (17 days)	Berland et al., 1976
8 spp.	12—8,000	LC_{50} (0.5-hr exposure plus 168-hr postexposure	Boney, 1971; Boney and Corner, 1959
5 spp.	60	LC_{100}	Ukeles, 1962
Laminaria hyperboria	2,500	LC_{100} (24 hr)	Hopkins and Kain, 1971
Coelenterata			
Eirene viridula	300	LC_{100} (3 hr)	Karbe, 1972
Echinodermata			
Acanthocidaris crassispina eggs	18—46	LC_{100} (48 hr)	Kobayshi, 1971
Asterias forbesi	10	LC_0 (168 hr)	Eisler and Hennekey, 1977
Asterias forbesi	20	LC_{50} (168 hr)	
Asterias forbesi	125	LC_{100} (168 hr)	
Annelida			
Neanthes arenaceodentata	22—100	LC_{50} (96 hr)	Reish et al., 1976
Neanthes arenaceodentata	17—90	LC_{50} (28 days)	
Capitella capitata	14—<100	LC_{50} (96 hr)	
Capitella capitata	100	LC_{50} (28 days)	
Nereis virens	25	LC_0 (168 hr)	Eisler and Hennekey, 1977
Nereis virens	60	LC_{50} (168 hr)	
Nereis virens	125	LC_{100} (168 hr)	
Ophryotrocha labronica	1,000	LC_{50} (0.5 hr)	Brown and Ahsanullah, 1971
Mollusca			
Mya arenaria	1	LC_0 (168 hr)	Eisler and Hennekey, 1977
Mya arenaria	4	LC_{50} (168 hr)	
Mya arenaria	30	LC_{100} (168 hr)	
Mercenaria mercenaria embryos	2.5	LC_0 (48 hr)	Calabrese and Nelson, 1974
Mercenaria mercenaria embryos	4.8	LC_{50} (48 hr)	
Mercenaria mercenaria embryos	7.5	LC_{100} (48 hr)	
Crassostrea virginica embryos	1.0	LC_0 (48 hr)	Calabrese et al., 1973
Crassostrea virginica embryos	5.6	LC_{50} (48 hr)	
Crassostrea virginica embryos	8.0	LC_{100} (48 hr)	

(Table continues on following page.)

Table 1 (Continued)

Species	Concentration	Effect	Reference
Mollusca (continued)			
Crassostrea virginica	50	LC_{50} (19 days)	Kopfler, 1974
Argopecten irradians	89	LC_{50} (96 hr)	Nelson et al., 1976
Nassarius obsoletus	100	LC_0 (168 hr)	Eisler and Hennekey, 1977
Nassarius obsoletus	700	LC_{50} (168 hr)	
Nassarius obsoletus	5,000	LC_{100} (168 hr)	
Congeria leucophaeta	1,000	LC_{20} (48 hr)	Dorn, 1974
Congeria leucophaeta	10,000	LC_{100} (48 hr)	
Cardium edule	10,000	LC_{50} (48 hr)	Portmann, 1968
Rangia cuneata	6,300–40,000	LC_{50} (48 hr)	Olson and Harrel, 1973
Crustacea			
Tigriopus japonicus	6	LC_{100} (72 hr)	D'Agostino and Finney, 1974
Paneus setiferus postlarvae	17	LC_{50} (96 hr)	Green et al., 1976
Pagurus longicarpus	10	LC_0 (168 hr)	Eisler and Hennekey, 1977
Pagurus longicarpus	50	LC_{50} (168 hr)	
Pagurus longicarpus	125	LC_{100} (168 hr)	
Petrolisthes armatus	50–64	LC_{50} (96 hr)	Roesijadi et al., 1974
Sesarma sp.	60	LC_{50} (24 hr)	Okubo and Okubo, 1962
Pandalus montague	100	LC_{50} (48 hr)	Portmann, 1968
Uca pugilator larvae	180	LC_{100} (24 hr)	DeCoursey and Vernberg, 1972
Uca pugilator	1,000	LC_{60} (28 days)	Weis, 1976
Nitocra spinopes	600	LC_{50} (24 hr)	Barnes and Stanbury, 1948
Carcinus maenus	1,000	LC_{50} (48 hr)	Portmann, 1968
Balanus balanoides	1,000	LC_{90} (48 hr)	Clarke, 1947
Crangon crangon	6,000	LC_{50} (48 hr)	Portmann, 1968
Artemia salina	1,000	LC_{50} (25 hr)	Brown and Ahsanullah, 1971
Artemia salina	21,000–50,000	LC_{50} (24 hr)	Okubo and Okubo, 1962

(Table continues on following page.)

fect	Reference
19 days)	Kopfler, 1974
96 hr)	Nelson et al., 1976
68 hr)	Eisler and Hennekey, 1977
(168 hr)	
(168 hr)	
(48 hr)	Dorn, 1974
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(48 hr)	Portmann, 1968
(48 hr)	Olson and Harrel, 1973
(72 hr)	D'Agostino and Finney, 1974
(96 hr)	Green et al., 1976
168 hr)	Eisler and Hennekey, 1977
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(96 hr)	Roesijadi et al., 1974
(24 hr)	Okubo and Okubo, 1962
(48 hr)	Portmann, 1968
(24 hr)	DeCoursey and Vernberg, 1972
(23 days)	Weis, 1976
(24 hr)	Barnes and Stanbury, 1948
(48 hr)	Portmann, 1968
(48 hr)	Clarke, 1947
(48 hr)	Portmann, 1968
(25 hr)	Brown and Ahsanullah, 1971
(24 hr)	Okubo and Okubo, 1962

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Table 1 (Continued)

Species	Concentration	Effect	Reference
Teleosts			
Mugil cephalus	500	LC_{58} (3 hr)	Middaugh and Rose, 1974
Mugil cephalus	50	LC_0 (3 hr)	
Fundulus heteroclitus	100	LC_0 (168 hr)	Eisler and Hennekey, 1977
Fundulus heteroclitus	800	LC_{50} (168 hr)	
Fundulus heteroclitus	1,000	LC_{100} (168 hr)	
Fundulus heteroclitus	1,000	LC_{100} (168 hr)	Weis and Weis, 1976
Fundulus heteroclitus	100	LC_{100} (264 hr)	
Fundulus heteroclitus	860	LC_0 (96 hr)	Klaunig, Koepp, and McCormick, 1975
Fundulus heteroclitus	2,000	LC_{50} (96 hr)	
Fundulus heteroclitus	4,600	LC_{100} (96 hr)	

*Concentrations are in micrograms per liter (ppb) of total mercury added at start of the experiment which were lethal to specified percentage within the indicated time frame.

and Stanbury, 1948; Corner and Sparrow, 1956; Gray, 1974; Gray and Ventilla, 1973; Hunter, 1949; Lask et al., 1971); anion concentration in the medium (Amend, Yasutake, and Morgan, 1969; Lask et al., 1971); complexing agents in solution (Boney and Corner, 1959; Eisler et al., 1972; Nishikawa and Tabata, 1969; Tabata and Nishikawa, 1969); presence of surface active agents (Calamari and Marchetti, 1973; Eisler et al., 1972); rate at which mercury was administered (Corner and Rigler, 1957; Gillespie, 1972; Horn and Katz, 1946); temperature of the water (Amend, Yasutake, and Morgan, 1969; Ballard and Oliff, 1969; Boney and Corner, 1959; Clemens and Sneed, 1958; Jones, 1973; Lask et al., 1971; MacLeod and Pessah, 1973; Portmann, 1968; Rodgers et al., 1951; Vernberg, DeCoursey, and O'Hara, 1974; Vernberg and O'Hara, 1972; Vernberg and Vernberg, 1972); seasonal differences, as distinct from temperature (Vernberg, DeCoursey, and O'Hara, 1974); dissolved oxygen content (Amend, Yasutake, and Morgan, 1969); pH (Lask et al., 1971); and salinity of the medium (Binet and Nicolle, 1940; Boetius, 1960; Hunter, 1949; Jones, 1973; 1975; Olson and Harrel, 1973; Pyefinch and Mott, 1948; Roesijadi et al., 1974; Schneider, 1972; Vernberg, DeCoursey, and O'Hara, 1974; Vernberg and Vernberg, 1972). Biological factors modifying the mercury survival response of marine organisms include: age of test organisms (Connor, 1972; Green et al., 1976; Okubo and Okubo, 1962; Portmann, 1968; Reish et al., 1976; Vernberg, DeCoursey, and O'Hara, 1974; Wilson and

Connor, 1971); sex (Vernberg, DeCoursey, and O'Hara, 1974; Vernberg and Vernberg, 1972); general health of animal (Portmann, 1968; Shealy and Sandifer, 1975); previous exposure to organic compounds (Boney and Corner, 1959); or to mercury (Corner and Sparrow, 1956; Foster and Olson, 1951; Gillespie, 1972; Green et al., 1976); moulting stage of crustaceans (Wilson and Connor, 1971); and presence of parasites (Clemens and Sneed, 1958).

It was not possible to document a specific trend for any individual modifier. Some studies showed relatively short survival times at comparatively high temperatures, and others demonstrated unchanged survival times for some species at a Δt of 10°C or more. Most studies, however, showed that previous exposure to mercury salts was in some way associated with increased sensitivity to mercury and other stressors; shrimp were an exception. Pre-exposure of shrimp for 57 days to 1.0 μg Hg/liter did not affect LC_{50} values (Green et al., 1976). There was general agreement that organo-mercury compounds were more toxic than inorganic compounds; that chelating agents in combination with mercury salts produced less-than-additive toxicity but other compounds, especially salts of copper, lead, or zinc act synergistically to increase toxicity; and that salinity stress, especially abnormally low salinities, reduced significantly the survival time of mercury-exposed organisms. The evidence suggests that species adapted to a fluctuating estuarine environment could be more vulnerable to the added stresses of heavy metal pollution, including mercury, than species inhabiting more uniformly stable environments (Jones, 1973).

Sublethal and Latent Effects

Mercury adversely affects various metabolic processes essential to normal growth, development, reproduction, and general well-being of marine and estuarine biota. Typically, metabolic effects are manifested at mercury concentrations that are orders of magnitude below those producing death. Morphological variations, for example, occur in dinoflagellates at 1.0 μg Hg/liter (Kayser, 1976), and teratogenic effects, such as the abnormal growth of multiple eyes and tentacles, are found in marine gastropods exposed only as embryos for 3 to 24 days to 10.0 μg Hg/liter (Reinhart and Myers, 1975). Exposure of shrimp larvae for 48 hr to high sublethal mercury levels is associated with reduced survival at post larval stage, delayed molting, extended development time, and morphological deformities (Shealy and Sandifer, 1975). At 1/100th of the lethal concentration, mercury disrupts avoidance-learning responses in fish (Weir and Hine, 1970), and, in fact, mercury is more effective in reducing these conditioned

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responses than are salts of arsenic, lead, or selenium (Weir and Hine, 1970). Altered feeding behavior and appetite reduction during and after exposure to mercury salts are documented for teleosts (Klaunig, Koepp, and McCormick, 1975; Rodgers et al., 1951; Salzinger et al., 1973) and mussels (Dorn, 1976).

Mercury-induced growth inhibition is reported for several marine species. The sediment-living, ciliate protozoan *Cristigera* exhibited reduction in growth during immersion in 2.5 to 5.0 μg Hg/liter (Gray, 1974; Gray and Ventilla, 1973); synergistic effects of Hg—Cu and Hg—Pb mixtures on *Cristigera* growth were also recorded (Gray and Ventilla, 1971). Growth of the colonial, marine hydroid *Campanularia flexuosa* was depressed during immersion for 11 days in 1.6 to 1.7 μg Hg/liter; however, growth stimulation occurred in 1.0 μg Hg/liter between days 3 and 7 (Stebbing, 1976). Kelps and other species of algae exhibited growth inhibition during immersion in < 5.0 to 350.0 μg Hg/liter (Berland et al., 1976; Boney, 1971; Clendenning and North, 1959; Kayser, 1976; Sick and Windom, 1975); similar results were observed for various bacteria (Ben-Bassat et al., 1972). Photosynthesis was reduced in several species of marine alga during immersion in 0.06 to 10.0 μg Hg/liter; pronounced effects were consistently induced by organomercury compounds at concentrations of 1.0 μg/liter (Harriss, White, and MacFarlane, 1970; Hopkins and Kain, 1971; Nuzzi, 1972). Recovery of algae to control growth levels after initial mercury-induced inhibition was also reported (Davies, 1974).

Mercury disrupts various reproductive processes of aquatic organisms, including fecundity, egg deposition, and hatching frequency. Concentrations between 32 and 92 μg Hg/liter seriously impair development of sea urchin embryos (Okubo and Okubo, 1962; Waterman, 1937); higher concentrations (1,000 μg/liter) were necessary to produce a similar effect on marine protozoa (Persoone and Uyttersprot, 1975). Flagellar contractility of sea urchin sperm was inhibited during immersion in 20 to 200 μg Hg/liter (Young and Nelson, 1974); however, mercury was exceeded in this effect by Cd, Cu, Zn, and Fe (Morisawa and Mohri, 1974). Teleost zygotes fertilized and incubated in water containing at least 0.2 μg Hg/liter exhibit a reduced hatching rate (Kihlstrom, Lundberg, and Hulth, 1971). The number of teleost eggs deposited decreases when the concentration of organomercury compounds is 1.0 μg Hg/liter or greater (Kihlstrom, Lundberg, and Hulth, 1971). Eggs fertilized in mercury-free water and then allowed to develop under various mercury regimes exhibited increased hatch at 10.0 μg Hg/liter but reduced or negligible hatch at 50.0 μg Hg/liter (Kihlstrom and Hulth,

1972). Mercury also inhibits zoospore activity and subsequent development of sporangia in marine fungi (Schneider, 1972).

Mercury interferes with enzyme production or activity in marine teleosts, including brain cholinesterase (Abou-Donia and Menzel, 1967), blood transaminases and dehydrogenases (Christensen, 1971), hemopoietic Na—K—ATPase activities (Renfro et al., 1974), and liver aminolevulinate dehydrases and other enzymes (Jackim, 1973; Jackim, Hamlin, and Sonis, 1970). Low levels, 5.0 μg Hg/liter, increased allantoise formation in marine annelids, but higher levels prevent formation (May and Brown, 1973). Enzyme activities may have potential as an early warning system of mercury-induced stress. For example, a concentration of 0.17 μg Hg/liter, one-tenth the level causing growth inhibition of marine hydroids, inhibited the lysosomal hydrolase staining reaction in that group (Moore and Stebbing, 1976).

Histopathological damage was observed among marine teleosts subjected to 500 μg Hg/liter; damage was most severe in olfactory organs and in the lateral line system (Gardner, 1975). An observed dose-related increase in mantle tentacle epithelial cells of mercury-treated clams (Fowler, Wolfe, and Hettler, 1975) suggests that histological studies might also make it possible to predict impending mercury stress.

Mercury affects oxygen consumption and ventilation rates of aquatic animals. Increased respiration of the mollusc *Congeria leucophaeta* was observed during immersion in 10.0 μg Hg/liter for 48 hr (Dorn, 1974). Concentrations of 3.0 μg Hg/liter caused a significant increase in "cough frequency" of trout, and this may alter oxygen consumption in that species (Drummond, Olson, and Batterman, 1974). Larvae of the fiddler crab *Uca pugilator* exposed to 1.8 μg Hg/liter for 24 hr showed a marked reduction in swimming ability and reduction in general metabolic processes, including oxygen consumption (DeCoursey and Vernberg, 1972). Marine teleosts exposed for 60 days to 10.0 μg Hg/liter exhibited elevated respiration rates, and, at 5.0 μg/liter, there were increases in plasma proteins and decreases in plasma osmolality (Calabrese et al., 1975).

Studies with antifouling compounds suggest that mercury is relatively ineffective in preventing metamorphosis of barnacles attached to glass plates (Clarke, 1947). Some mariculture studies have indicated that low concentrations of mercury salts for short periods have either no measurable effect or beneficial, therapeutic, or prophylactic applications (Green et al., 1976; Rodgers et al., 1951; Rucker and Amend, 1969). The preponderance of studies, however, seems to demonstrate that mercury adversely affects several vital life

processes of marine organisms at concentrations as low as 0.2 and 1.0 μg/liter. In addition to the deleterious effects previously listed on behavior, growth, reproduction, histology, respiration, and enzyme production, there are a variety of mercury-related effects that are imperfectly understood. These include increased mucous production in fish (Gardner, 1975; Lask et al., 1971); initiation of diuresis in amphipods (Lockwood and Inman, 1975); a rise in urine nitrogen levels of mercury-poisoned crabs (Corner, 1959); altered osmoregulatory ability of estuarine isopods (Jones, 1975); and negative phototaxy and increased operculate movements of teleosts (Klaunig, Koepp, and McCormick, 1975). Increased efficiency of mercury transport mechanisms has also been noted in crabs exposed at comparatively high temperatures (Vernberg and O'Hara, 1972).

Uptake, Retention, and Translocation

Rapid accumulation of mercury, especially organomercury compounds, by various species of marine biota, primarily teleosts and molluscs, is well documented (Bligh, 1972; Boulton and Hetling, 1972; Cunningham and Tripp, 1975a; 1975b; Davies, 1974; Fang, 1973; Fowler, Wolfe, and Hettler, 1975; Hannerz, 1968; Hasselrot, 1968; Hibiya and Oguri, 1961; Johnels et al., 1967; Kramer and Neidhart, 1975; Lask et al., 1971; Laumond et al., 1973; MacLeod and Pessah, 1973; McKone et al., 1971; Middaugh and Rose, 1974; Nelson et al., 1976; Olson, Bergman, and Fromm, 1973; Pentreath, 1976a; 1976b; Rucker and Amend, 1969). Bioaccumulation of mercury and its compounds from seawater can be modified by many factors. These include chemical form of mercury administered (Cunningham and Tripp, 1975a; Hannerz, 1968; Kramer and Neidhart, 1975; Pentreath, 1976a; 1976b); mode of administration (Jarvenpaa, Tillander, and Miettinen, 1970); presence of complexing agents in medium (Kramer and Neidhart, 1975); initial concentration (Kramer and Neidhart, 1975; Sick and Windom, 1975); exposure time (Sick and Windom, 1975); presence of selenium (Fowler and Benayoun, 1976); salinity of medium (Vernberg, DeCoursey, and O'Hara, 1974); water temperature (Cunningham and Tripp, 1975b; Vernberg, DeCoursey, and O'Hara, 1974); age of organism (Beckett and Freeman, 1974; Cunningham and Tripp, 1975b; Glooschenko, 1969; Hannerz, 1968); biological surface area (Sick and Windom, 1975); variability in detoxication mechanisms (Davies, 1976); sexual condition (Cunningham and Tripp, 1975b); tissue specificity (Cunningham and Tripp, 1975a; Fowler, Wolfe, and Hettler, 1975; Hannerz, 1968; Pentreath, 1976a; 1976b; Vernberg, DeCoursey, and O'Hara, 1974); presence of mercury-resistant strains of bacteria

(Colwell and Nelson, 1975; Colwell et al., 1976); and accumulation after death (Glooschenko, 1969). Two studies, both on adult American oysters, *Crassostrea virginica*, are worth emphasizing. Cunningham and Tripp (1973) held oysters in seawater containing 10 μg Hg/liter as mercuric acetate. After 45 days the whole-body mercury concentration of exposed animals was 28,000 μg/kg wet weight. The mercury concentration in control oysters was always less than 20 μg/kg wet weight. The mercury concentration in exposed oysters dropped to 18,000 μg/kg by day 60, probably because of spawning. At day 60, oysters were transferred to mercury-free seawater for 160 days. During the first 18 days, levels declined to 15,000 μg Hg/kg, but thereafter no further decline occurred. It was concluded that oysters can concentrate 10 μg Hg/liter by a factor of 2800 and that total self-purification was not achieved over a 6-month cleansing period. Kopfler (1974) found that continuous exposure to 1 μg Hg/liter in any of the three mercury compounds tested caused oysters to concentrate mercury rapidly in their tissues far in excess of 0.5 mg/kg wet weight, i.e., at levels potentially hazardous to humans ingesting these oysters, according to the action guideline established by the Food and Drug Administration. Details of Kopfler's study follow:

> Accumulation of mercury compounds by oysters was determined in two experiments, each utilizing three groups of 100 adult oysters. In the first experiment, conducted between 0 and 10°C, mercury levels were maintained at 50 μg/l with flow rate adjusted to one liter per oyster per hour. In the second experiment, mercury levels were reduced to one μg/l, the water temperature varied between 25 and 35°C, and flow rates maintained at 2 l/oyster/hr because of the increased temperature. Controls were maintained for each study. In the first experiment, the administration of organic mercury compounds was terminated after 19 days because many of the oysters in the groups receiving salts of either methylmercury or phenylmercury were dead or moribund. Oysters classified as moribund exhibited slow, incomplete valve closure when disturbed. When oysters which survived 19 days of exposure to methylmercury and phenylmercury were placed in flowing seawater, about half in each group died within a week with all oysters in both groups dead within 14 days. Oysters exposed to inorganic mercuric chloride exhibited no apparent ill effects over a 42-day period of exposure to 50 μg/l of Hg. Mean mercury levels in experimentals after one week was 1000X over controls. Mercury values in experimental oysters at 7 days, in mg Hg/kg wet wt, ranged between 15 and 25; for controls this was 0.02. Copper and zinc levels were depressed in flesh from all three groups of oysters after one week exposure to mercury; in the two groups exposed to organomercury, Cu and Zn declined over the 19-day exposure. Copper and zinc levels in the oysters exposed to mercuric chloride began to increase during the third week and continued to do so until they were essentially the same as control values. In the second experiment methyl- and phenylmercury were concentrated

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to essentially the same degree, while inorganic mercury was concentrated about 4 times less over a 74-day exposure. For organomercury compounds, values in mg Hg/kg wet weight were about 10 at 20 days and 30 at 60 days (a final concentration factor of about 30,000). For inorganic mercuric chloride, values were about 2 at 20 days and about 10 at 60 days.

Mercury is also accumulated from seawater by other marine organisms. In radiotracer studies, clams (*Tapes decussatus*) contained 10 times more ^{203}Hg per unit weight than the medium within 24 hr (Unlu, Heyraud, and Keckes, 1972). Two species of marine alga, *Chaetoceros galvestonensis* and *Phaeodactylum tricornutum*, contained 7.4 and 2.4 g/kg of mercury, respectively, when cultured in media containing 100 μg Hg/liter (*Chaetoceros*) or 50 μg Hg/liter (*Phaeodactylum*) (Hannan et al., 1973).

Mercury can also be accumulated through food webs. Bacteria have a demonstrable effect on mercury accumulation in food chains that include filter feeders. Thus whole oyster accumulation of mercury more than doubled in the presence of mercury-resistant strains of *Pseudomonas* spp. (Colwell and Nelson, 1975; Colwell et al. 1976; Sayler, Nelson, and Colwell, 1975). Sarcophagous flies feeding on mercury-contaminated fish muscle accumulated 3 times more mercury than their food; this was especially pronounced when the fish flesh contained > 0.5 mg Hg/kg wet weight (Nuorteva and Hasanen, 1972). A similar pattern was noted among wild fish feeding on mercury-contaminated fingerlings (Rucker and Amend, 1969). In the food chain algae-detritus—worm—fish (prey)—fish (predator), mercury had a long biological half-life, > 1000 days in predatory fish compared with about 55 days in prey fish (Huckabee and Blaylock, 1972). The transfer efficiency of inorganic mercury from prey to predator fish was about 40% but from worm to prey fish, only 12%; worms assimilated 60% of the inorganic mercury contained in the algae-detritus. Huckabee and Blaylock concluded that food-chain uptake can account for a significant percentage of the mercury body burden in fish. In an algae-to-copepod food link, however, the crustacea showed no impairment of egg laying or egg development and no retention of mercury in tissues, eggs, or feces (Parrish and Carr, 1976). No progressive mercury concentration was observed in a clam-to-eel transfer (Tsuruga, 1963). When dogfish meal containing up to 2.3 mg total Hg/kg, of which 1.9 mg/kg was methyl mercury, replaced low-mercury fish rations used in salmon culture, the flesh of the salmon contained > 0.5 mg/kg within 240 days (Spinelli and Mahnken, 1976). When dogfish meal comprised less than half the diet, however, mercury levels in salmon flesh were < 0.5 mg/kg. Similar results were observed among sablefish fed dogfish meal (Kennedy and Smith, 1972).

Skeletal muscle of teleosts appears to function as a reservoir for methyl mercury (Giblin and Massaro, 1973; Johnels et al., 1967; MacLeod and Pessah, 1973; Middaugh and Rose, 1974; Pentreath, 1976b; Weisbart, 1973). Maximum concentration factors of radiomercury were reached in skeletal muscle, brain, and lens after 34, 56, and >90 days, respectively; maximum values were reached in most other tissues and organs in about 7 days (Giblin and Massaro, 1973). One pathway by which anadromous fishes accumulate mercury from a medium is through the gills; up to 90% of the mercury taken up on the gills is subsequently bound to erythrocytes within 40 min (Olson, Bergman, and Fromm, 1973; Olson and Fromm, 1973).

The retention time of mercury in marine organisms depends on several factors. Weisbart (1973) found that the gill, heart, and swim-bladder tissues of fish lost mercury at rates faster than the whole organism, but some tissues, including brain, liver, muscle, and kidney, showed no significant decrease with time. In clams radiomercury seems to have a biological half-life of 10 days if accumulated via the food chain but only 5 days if taken up from the water (Unlu, Heyraud, and Keckes, 1972). Retention times of ^{203}Hg in most marine species were comparatively lengthy, ranging from 267 days for the fish *Serranus scriba* to 1000 days for mussels, *Mytilus galloprovincialis* (Miettinen, Heyraud, and Keckes, 1972; Miettinen et al., 1969; 1972). Miettinen and coworkers found that phylogenetically related species follow a similar pattern of methyl mercury excretion, with biological half-life depending on water temperature and mode of entry into organisms; it is longer after intramuscular injection than after peroral administration. There is some evidence that mercury excretion rates follow a biphasic or polyphasic pattern (Burrows and Krenkel, 1973; Giblin and Massaro, 1973; Tillander, Miettinen, and Koivisto, 1972; Weisbart, 1973). Tillander, Miettinen, and Koivisto found that, in seals, *Pusa hispada*, the fastest excreted component took 20 days for 50% elimination and the slowest excreted mercury fraction, which comprised 45% of all mercury, 500 days for 50% elimination.

Field Investigations

Residues in Water and Sediments

Mercury levels in coastal seawater from various locations ranged from 0.021 to 0.078 μg/liter (Dehlinger et al., 1973); concentrations were lower in continental slope water, ranging from 0.010 to 0.041 μg/liter. Mercury levels in water were several orders of

magnitude higher in the vicinity of chlor—alkali plants and similar industrial operations (Kazantzis, 1971). Mercury levels in sediments varied widely and tended to be elevated in the neighborhood of sewer outfalls (Klein and Goldberg, 1970), sludge disposal areas, and especially areas impacted by mercury wastes from industrial operations, such as Minamata Bay, Japan (Irukayama, 1967). Vucetic, Vernberg, and Anderson (1974) reported values ranging from 130 to 1500 μg/kg of mercury in sediments in the Adriatic Sea, and Williams and Weiss (1973) found 390 μg/kg in pelagic clays collected 430 km southeast of San Diego, Calif. It is reported that mercury in seawater exists almost entirely bound to suspended particles (Jernelöv et al., 1972), that the surface area of sediment granules is instrumental in determining final mercury content (Renzoni, Bacci, and Falciai, 1974), that conversion and transformations occur in the surface layer of the sediment or on suspended organic particles in the water (Dean, 1972; Fagerstrom and Jernelöv, 1972; Jernelöv et al., 1972), and, finally, that mercury-containing sediments would require many decades to purge themselves naturally to background levels (Langley, 1973).

Mercury—sediment—water interactions affect uptake by marine life. Bivalves can accumulate mercury directly from seawater, and molluscs had higher concentrations of the metal in turbulent waters than in clear waters (Raymont, 1972). Further, marine organisms feeding in direct contact with sediment have higher overall mercury levels than those feeding above the sediment—water interface (Klemmer, Unninayer, and Ukubo, 1976).

Residues in Biota

Table 2 summarizes the ranges of concentrations of mercury in field collections of marine flora and fauna. These data support the findings of other investigators (Cocoros, Cahn, and Siler, 1973; Huckabee and Blaylock, 1972; Jernelöv and Lann, 1971; Knauer and Martin, 1972; Skei, Saunders, and Price, 1976; Stickney et al., 1975), who demonstrated that the efficiency of mercury transfer through natural marine food chains among lower trophic levels is comparatively low. Higher trophic levels, such as fishes, fish-eating birds, and mammals, however, exhibit marked mercury amplification. The variability of concentrations is partly explainable in terms of collection locale; some field collections were taken from areas where human activities have raised the mercury content in the aquatic environment above natural levels, thus producing a significant increase in the mercury content of endemic fauna (Hearnden, 1970; Johnels et al., 1967; Kazantzis, 1971; Kleinert and Degurse, 1972;

TABLE 2

RANGE IN CONCENTRATIONS OF MERCURY (μg/kg wet weight) REPORTED FROM FIELD COLLECTIONS OF MARINE BIOTA

Sample	Concentration	Reference
Algae	40—2,000	Greig, Wenzloff, and Shelpuk, 1975; Haug, Melsom, and Omang, 1974; Leatherland and Burton, 1974; Lulic and Strohal, 1974
Higher aquatic plants	10—8,700	Gardner et al., 1975; Johnson and Braman, 1975; Lindberg and Harriss, 1974; Windom, 1973
Porifera	330—1,580	Leatherland and Burton, 1974; Williams and Weiss, 1973
Coelenterata	70—860	Leatherland and Burton, 1974; Leatherland et al., 1973
Tunicata	60—570	Leatherland and Burton, 1974; Leatherland et al., 1973
Echinodermata	280—400	Williams and Weiss, 1973
Annelida	350	Leatherland and Burton, 1974
Mollusca	1—4,440	Anderlini, 1974; Burton and Leatherland, 1971; Craig, 1967; Greig, Wenzloff, and Shelpuk, 1975; Hussain and Bleiler, 1973; Irukayama et al., 1962a; 1962b; Laumond et al., 1973; Leatherland and Burton, 1974; Lulic and Strohal, 1974; Stenner and Nickless, 1975; Thibaud, 1973; Vucetic, Vernberg, and Anderson, 1974; Williams and Weiss, 1973; Young, 1974
Crustacea	5—2,520	DeClerk, Vanderstoppen, and Vyncke, 1974; Eftekhari, 1975; Evans, Bails, and D'Itri, 1972; Greig, Wenzloff, and Shelpuk, 1975; Holden and Topping, 1972; Johnson and Braman, 1975; Leatherland et al., 1973; Raeder and Snekvik, 1949b; Skei, Saunders, and Price, 1976; Somayajulu and Rama, 1972; Stenner and Nickless, 1975; Vucetic, Vernberg, and Anderson, 1974; Williams and Weiss, 1973
Insecta	221—630	Kim, Chu, and Barron, 1974

(Table continues on following page.)

Renzoni, Bacci, and Falciai, 1974; Windom, Taylor, and Stickney, 1973; Wobeser et al., 1970; Zitko et al., 1971).

Inshore marine biota often contain higher mercury concentrations than the same or similar species collected offshore (Dehlinger et al., 1973; Jones, Jones, and Stewart, 1972; Westoo, 1969). In

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ted offshore (Dehlinger
72; Westoo, 1969). In

Table 2 (Continued)

Sample	Concentration	Reference
Elasmobranchs	24—2,080	Childs and Gaffke, 1973; Childs, Gaffke, and Crawford, 1973; Forrester, Ketchen, and Wong, 1972; Gardner et al., 1975; Leatherland et al., 1973; Peterson, Kalwe, and Sharp, 1973; Windom et al., 1973
Teleosts	10—6,300	Alexander et al., 1973; Arima and Umemoto, 1976; Barber, Vijayakumar, and Cross, 1972; Beasley, 1971; Beckett and Freeman, 1974; Benson et al., 1976; Childs and Gaffke, 1973; Cocoros, Cahn, and Siler, 1973; Cugurra and Maura, 1976; DeGoeij and Zegers, 1971; Establier, 1975a; Freeman and Horne, 1973; Gardner et al., 1975; Greig, Wenzloff, and Pearce, 1976; Greig, Wenzloff, and Shelpuk, 1975; Greve and Wit, 1971; Johnson and Braman, 1975; Kamps, Carr, and Miller, 1972; Leatherland and Burton, 1974; Lulic and Strohal, 1974; Peterson, Kalwe, and Sharp, 1973; Portmann, 1972; Raeder and Snekvik, 1949a; 1949b; Renzoni, Bacci, and Falciai, 1974; Shultz et al., 1976; Stenner and Nickless, 1975; Stickney et al., 1975; Suzuki, Miyama, and Toyama, 1973; Ui and Kitamura, 1971; Vucetic, Vernberg, and Anderson, 1974; Williams and Weiss, 1973; Windom et al., 1973
Birds	50—65,000	Benson et al., 1976; Berg et al., 1966; Koivusaari et al., 1976
Mammals	40—387,000	Anas, 1971; 1974; Buhler, Claeys, and Mate, 1975; Gaskin et al., 1972; 1973; 1974; Holden, 1975; Jones et al., 1976; Kim, Chu, and Barron, 1974; Koeman et al., 1973; Roberts, Heppleston, and Roberts, 1976; Robertson et al., 1972; Sergeant and Armstrong, 1973; Tillander, Miettinen, and Koivisto, 1972

Sweden, marine fish caught near shore often showed comparatively high methyl mercury levels, with many values in the range from 5,000 to 10,000 μg/kg wet weight (Ackefors, Lofroth, and Rosen, 1970; Westoo, 1969); however, levels above 1,000 μg/kg in Swedish fish are usually caused by industrial discharges of mercury compounds (Westoo, 1969).

Several observations can be made regarding mercury residues in teleosts collected in the field. First, mercury tends to concentrate in the edible flesh of finfish, with older fish containing more mercury per unit weight than young fish (Alexander et al., 1973; Barber, Vijayakumar, and Cross, 1972; Cross et al., 1973; Cumont et al., 1972; DeClerk, Vanderstoppen, and Vyncke, 1974; Evans, Bails, and D'Itri, 1972; Forrester, Ketchen, and Wong, 1972; Giblin and Massaro, 1973; Greichus, Greichus, and Emerick, 1973; Hannerz, 1968; Johnels and Westermark, 1969; Johnels et al., 1967; Kleinert and Degurse, 1972; Knight and Herring, 1972; Nuorteva and Hasanen, 1971; 1975; Peterson, Kalwe, and Sharp, 1973; Scott and Armstrong, 1972; Svansson, 1975; Taylor and Bright, 1973; Vermeer, 1972). Second, most of the mercury in fish flesh was in the organic form, primarily methyl mercury (Fukui et al., 1973; Kamps, Carr, and Miller, 1972; Koeman et al., 1973; Peterson, Kalwe, and Sharp, 1973; Rissanen, Erkama, and Miettinen, 1972; Westoo, 1966; 1969; 1973; Zitko et al., 1971). Third, levels of mercury in muscle from adult tunas, billfishes, sharks, and other carnivores were higher than those in young fishes with a shorter food chain; this indicates associations among predatory behavior, longevity, and mercury accumulation (Forrester, Ketchen, and Wong, 1972; Jernelöv, 1972; Peakall and Lovett, 1972; Peterson, Kalwe, and Sharp, 1973; Ratkowsky, Dix, and Wilson, 1975; Saila, 1971; Ui, 1972; Walter, June, and Brown, 1973). Finally, elevated levels of mercury in wide-ranging oceanic fish are not solely the consequence of anthropogenic activities but also result from natural concentrations as well (Greig, Wenzloff, and Pearce, 1976; Miller et al., 1972; Schultz et al., 1976).

Birds that feed on aquatic animals exhibit markedly increased concentrations of mercury in comparison with terrestrial raptorial species (Johnels and Westermark, 1969; Karppanen and Henriksson, 1970). Top trophic-level predators, such as cormorants and pelicans, demonstrated mercury concentration factors in their flesh of 14- and 6-fold, respectively, over prey fish (Greichus, Greichus, and Emerick, 1973). Mercury levels in aquatic and fish-eating birds were elevated in the vicinity of chlor—alkali plants where mercury is used as a catalyst (Fimreite, 1974; Fimreite et al., 1971); these differences were detectable up to 300 km from the chlor—alkali plant (Fimreite and Reynolds, 1973). Eggs of fish-eating birds, including herons and grebes, collected near mercury point-source discharges contained abnormally high levels of mercury; 29% of eggs contained more than 0.5 mg/kg wet weight, and 9% contained more than 1.0 mg/kg (Faber and Hickey, 1973). Seasonal variations in mercury levels

occur in livers of aquatic birds. Higher levels were observed in winter, when birds were exclusively estuarine, and drastically lower levels were observed in summer when birds migrate to inland Arctic and sub-Arctic breeding grounds (Parslow, 1973).

Diet is an important concentrating mechanism in marine mammals. Gray seals and harbor seals, which feed on large fish and cephalopods, contained 10 times more mercury than harp seals, which feed on small pelagic fish and crustacea (Sergeant and Armstrong, 1973). Age and tissue specificity also influence mercury residue levels. Liver from older sea lions contained more mercury per unit weight than that from younger specimens (Anas, 1974; Holden, 1975; Jones et al., 1976; Sergeant and Armstrong, 1973). Some adult seals found dead had high levels of mercury in the brain in comparison with levels in various species poisoned by exposure to mercury compounds (Koeman et al., 1973). Mercury and selenium concentration in livers of marine mammals seem to be positively correlated (Koeman et al., 1973; 1975; Martin et al., 1976). Koeman and co-workers (1973) suggested that selenium protects these species against mercury poisoning by completely binding to subcellular S sites, the presumed location of mercury's toxic action.

Three points are worth emphasizing at this juncture. First, mercury discharged into rivers, bays, or estuaries as metallic mercury, inorganic divalent mercury, phenyl mercury, or alkoxyalkylmercury can all be converted to methyl mercury compounds by natural processes (Jernelöv, 1969). Second, organomercury complexes are rapidly accumulated in tissues with high lipid content (Wood, 1973). Finally, mercury-resistant strains of bacteria which have been developed or discovered may have application in mercury mobilization or fixation from mercury-contaminated waters to the extent that polluted areas become innocuous (Colwell and Nelson, 1975; Nelson et al., 1973; Vosjan and Van der Hoek, 1972).

Minamata

Any review of mercury hazards in the marine environment should include the Minamata Bay incident in southwestern Kyushu, Japan. This extensively documented case (Fujiki, 1963; Irukayama, 1967; Irukayama et al., 1961; 1962a; 1962b; Kiyoura, 1963; Kurland, Faro, and Siedler, 1960; Matida and Kumada, 1969; Matida et al., 1972; Takevchi, 1972; Tsubaki et al., 1967) reveals effects upon man of chronic discharges of low-level methyl mercury wastes into coastal waters. The source of mercury was waste discharged from an acetaldehyde plant into Minamata Bay beginning in 1952.

Several years later, the mercury levels in sediments near the plant outfall were about 2010 mg/kg wet weight; this decreased sharply with distance from the plant. Sediments in the bay contained between 0.4 and 3.4 mg Hg/kg wet weight. Concentrations of mercury in fish, shellfish, and other food decreased with increasing distance from the point of effluence and appeared to reflect sediment mercury levels. Late in 1953, a severe neurological disorder was recognized among inhabitants of the Minamata Bay region. By 1956, the outbreak had reached epidemic proportions; 111 cases of poisoning were found by the end of 1960; and 41 deaths were reported as of August 1965. The poisoning was caused by eating fish and shellfish from Minamata Bay and the neighboring sea, except for 19 congenital cases in children born of mothers who had eaten the same diet. In addition to humans, cats and water fowl living near the bay succumbed to the disease. Experimental cats and rats fed fish and shellfish collected from the bay developed the same symptoms as animals spontaneously affected. Symptoms included cerebellar ataxia, constriction of visual fields, dysarthria, and, in congenital cases, disturbance of physical and mental development. Pathological findings included regressive changes in the cerebellum and the cerebral cortexes. The clinico-pathological features resembled alkyl mercury poisoning. Abnormal mercury content (i.e., >30.0 mg Hg/kg wet weight) was measured in fish, shellfish, and muds from the bay and in organs of necropsied humans and cats that had succumbed to the disease. Years after the waste-discharge situation was corrected, fish and shellfish still contained levels of mercury hazardous to human health. Mercury levels in Minamata fish and shellfish were significantly higher than levels shown in Table 2 for teleosts and molluscs; note also that the high levels shown for mammals in Table 2 are from liver.

Potential Health Hazards

The dangerous mercury level in human blood is about 2000 ng/g; the normal background level in about 80% of people without occupational exposure to mercury is <5 ng/g (Saha, 1972). Since methyl mercury has a biological half-life of 70 to 200 days in man (Lofroth, 1970), it is not unexpected that mutagenic and teratogenic effects of mercury have been reported at levels well below those associated with acute poisoning (Kazantzis, 1971; Keckes and Miettinen, 1972).

The findings of elevated blood mercury levels, increased human chromosome breakage, and easy passage of mercury through placental membranes among people who regularly eat fish containing

1 to 17 mg Hg/kg wet weight suggest to some health authorities that the currently accepted level in the United States [0.5 mg Hg/kg wet weight (500 μg/kg)] does not have an appreciable level of safety for pregnant women (Establier, 1975b; Peakall and Lovett, 1972; Skerfving, Hansson, and Lindstem, 1970). In Sweden, the official limit for mercury in fish is 1.0 mg/kg wet flesh. Human risk in consumption of mercury-contaminated fish within this guideline is considered negligible in that country (Berglund and Berlin, 1969); however, some Scandinavian scientists recommend that fish consumption should be limited to one meal a week (Lofroth, 1970).

Selected Studies

Mercury retention by teleost species is variable. In one study, when northern pike from a lake that was heavily contaminated with mercury were transplanted to waters relatively free of mercury, only 30% of the mercury in their muscle was eliminated in 1 year (Lockhart et al., 1972). A study by Amend (1970) of juvenile sockeye salmon indicated that fish treated repeatedly with mercurials during their freshwater phase accumulated and retained high levels of mercury for several months, but, after 4 years at sea, the returning salmon contained normal levels of mercury in all tissues examined.

A number of investigators report that mercury from point-source discharges, including sewer outfalls and chlor—alkali plants, was taken up by sediments; sediment levels were eventually reflected by an increased mercury content of epibenthic fauna (Dehlinger et al., 1973; Hoggins and Brooks, 1973; Klein and Goldberg, 1970; Klemmer, Luoma, and Lau, 1973; Parsons, Bawden, and Heath, 1973; Takevchi, 1972). Analysis of the effluent from the Hyperion sewer outfall in Los Angeles showed a mercury content slightly below 1 μg/liter (Klein and Goldberg, 1970). Concentrations of mercury in sediments near this outfall were as high as 820 μg/kg but decreased with increasing distance from the outfall; mercury levels in epibenthic fauna, including crabs, whelks, and scallops, were also highest near the discharge and lowest tens of kilometers away.

A mercury budget for estuaries along the Georgia coast indicates that the dominant salt-marsh plant, *Spartina alterniflora*, exerts a strong control on mercury migration (Gardner et al., 1975; Windom et al., 1976; Windom, 1973). Mercury entered the estuary primarily in solution, delivering ~1.5 mg annually to each square meter of salt marsh. Annual uptake of mercury by *S. alterniflora* alone was about 0.7 mg/m^2 salt marsh. Mangrove vegetation plays a similarly important role in mercury cycling in the Florida everglades (Lindberg and Harriss, 1974; Tripp and Harriss, 1976).

RECOMMENDATIONS

On the basis of these studies, it appears that establishing marine mercury-contamination standards requires abatement of existing discharges, development of technologies to decontaminate polluted waterways, monitoring criteria for protecting human health, and research to modify existing standards, as well as to predict impending mercury stress in marine ecosystems. Specific recommendations are listed.

1. Prohibit the sale, manufacture, and use of methyl mercury compounds. All evidence presented to date implicates methyl mercury as the most toxic chemical form of mercury. Its demonstrated deleterious effects to marine biota and human health mandate passage of legislation banning all environmental uses of methyl mercury compounds. In the decade since Sweden prohibited agricultural use of methyl mercury for seed coatings and pesticides, bird populations have begun to recover and the amounts of mercury in many types of food have decreased (Johnels and Westermark, 1969).

2. Restrict use of other mercurials. Investigations are presently in progress in several countries on the geochemistry and biogeochemistry of mercury, but much remains to be learned about its distribution, its conversion from one form to another, and its transport on a global basis. It is well known, however, that inorganic mercury compounds can be converted in nature to organomercury compounds, which are more readily transported and accumulated in tissues. Accordingly, legislation is needed which will curtail environmental use of all mercury compounds, especially organomercurials.

3. Develop alternative technologies. About 90% of the mercury consumed annually is used for industrial purposes. Mercury is used as a catalyst in chlor–alkali plants, as a slimicide in pulp and paper mills, as a chemical reagent in the plastics industry, in pharmaceuticals and paints, in power plants in special heat engines, in metal-refinement operations by amalgamation, and in the manufacture of electric switches, batteries, and lamps. Chlor–alkali plants probably represent the greatest point-source hazard at this time; typical plants contain more than 500,000 kg of liquid mercury and discharge several kilograms daily to the environment. In the absence of better controls, this loss could be averted by developing a new technology that precludes mercury compounds. This technology is urgently required and should be awarded the highest priority.

4. Remove mercury from contaminated waterways. Thousands of kilometers of our inland waterways and some coastal areas are

contaminated with mercury to the extent that fish and other organisms taken from these waters are unsafe to eat (Anonymous, 1971). This process shows little sign of abatement. Accordingly, high priority should be given to research and development technology to remove mercury from existing contaminated areas. These could include bacterial strains and other methods to solubilize mercury from sediments, with subsequent removal and reclamation from waterways via biological or abiotic vectors. One interim alternative would be to feed mercury-contaminated scrapfish and seafoods as partial diet supplements to migratory anadromous fishes. Removal by dredging of mercury-contaminated sediments and storage at designated locations is another alternative, but numerous problems are associated with this approach as currently practiced.

5. Develop monitoring criteria. Preventing mercury feedback into human diets is of paramount importance. To achieve this goal, we must refine existing mercury-residue standards for waste waters, sediments, and biota, with due consideration for geographical location and economic aspects of the resource.

It is emphasized at this time that mercury in marine waters is usually bound to bottom materials, proteinaceous matter, plankton, and higher organisms. Even in water heavily polluted by mercury discharges, only a fraction is found free in solution, except near the point of discharge. The best available evidence indicates that total mercury in waste waters, dissolved plus particulate, emanating from the pipe terminus should not exceed 1.0 μg/liter; however, I believe two additional restrictions on pipe terminus concentrations are warranted to achieve a reasonable margin of safety. First, outfall volume should not exceed one-fiftieth of the mean daily waterflow of the receiving water. Second, the receiving waters upstream of the terminus (or in direction of positive tidal flow, if applicable) should contain less than 0.02 μg/liter total mercury, dissolved plus particulate. The final recommended value of 0.0392 μg total Hg/liter after mixing is about 2.5 times lower than the currently recommended value of less than or equal to 0.10 μg/liter proposed by the National Academy of Sciences (1973); however, the 0.10 μg/liter does not consider mixing processes.

Because benthic animals sometimes reflect the mercury levels of their sediment substrate, it is recommended that sediments in outfall environs should not exceed 500 μg total Hg/kg. This value is less than the 750 μg/kg set by the Environmental Protection Agency (1977) for solid wastes disposed offshore.

For biological monitoring, two groups of indicator organisms are recommended, filter-feeding bivalve molluscs and piscivorous fishes.

Total mercury levels in soft tissues from bivalves or fish axial musculature should not exceed 500 μg/kg wet weight (0.5 ppm). This is the current limit in comestibles set by the Food and Drug Administration. Until additional evidence becomes available it is also suggested that expectant mothers limit to 200 g/week their dietary intake of seafood items containing total mercury concentrations in the 0.3- to 0.5-ppm range.

6. Conduct research. One of the highest priorities should be assigned to refining the current analytical chemistry methods for detecting mercury. Specifically, procedures must be developed for accurate, precise, and inexpensive determination of mercury at concentrations as low as 0.02 μg/liter.

An equally high priority could be assigned to laboratory studies on lethal, sublethal, and latent effects of mercurials on selected marine indicator species during lifetime exposure. These data do not exist at present. Tests must be conducted in flowing seawater and should, at a minimum, accurately assess mercury effects on survival, growth, behavior, reproduction, accumulation, and histopathology. Results of these studies would provide a basis for a more reasonable assessment of safe levels of mercury in water and would probably result in some modification of the proposed 0.0392 μg/liter value recommended here.

Field and laboratory investigations of interaction effects of mercury with other constituents in the environment should be conducted. The significance of the apparent protective effect of selenium in marine mammals is a case in point. At present, data on field studies of mercury cycling and effects of mixed wastes containing mercury on community structure are scarce or absent. These studies should be conducted, with a major effort being assigned to roles played by aquatic mammals, fish-eating birds, predatory fishes, higher plants, and mercury-resistant strains of bacteria. Establishment of a nation-wide monitoring program on mercury levels in coastal sediments appears to be a necessary prerequisite for identifying heavily contaminated areas and selecting appropriate sites for field investigations.

One of the most important research needs is to develop an early warning system in selected indicator organisms that will forecast generalized stress among marine communities, with emphasis on response parameters peculiar to mercury-induced stress. Within the past decade, multiparametric approaches have been used extensively by industrial toxicologists and others who work with poisonous substances; however, application of these approaches to marine communities is rare and difficult. Typically, mercury-induced stress

would be evident long before physiological or morphological damage occurs, would be manifested at the cellular or subcellular level, and, if allowed to remain unabated, would ultimately cause death or large-scale changes in community structure. Biochemical responses are probably most sensitive at present, but behavioral and histological responses should not be discounted as possible early indicators.

7. Apply recommendations to other contaminants. With minor modifications, the preceding recommendations could become protocols for other heavy metals in coastal ecosystems and, with additional modifications, could be useful in abatement, decontamination, and research programs now in progress on many groups of synthetic chemicals routinely discharged into the marine environment.

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